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REVIEW

Measles vaccination: Threat from related veterinary viruses and need for continued vaccination post measles eradication

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ABSTRACT

Measles virus (MV) is the only human virus within the morbillivirus genus of the *Paramyxoviridae*. The veterinary members are canine distemper virus (CDV), peste des petits ruminants virus (PPRV), Rinderpest Virus (RPV) as well as the marine morbilliviruses phocine distemper virus (PDV), dolphin morbillivirus (DMV) and porpoise morbillivirus (PMV). Morbilliviruses have a severe impact on humans and animal species. They confer diseases which have contributed to morbidity and mortality of the population on a global scale. There is substantial evidence from both natural and experimental infections that morbilliviruses can readily cross species barriers. Of most concern with regard to zoonosis is the more recently reported fatal infection of primates in Japan and China with strains of CDV which have adapted to this host. The close genetic relationship, shared cell entry receptors and similar pathogenesis between the morbilliviruses highlights the potential consequences of complete withdrawal of MV vaccination after eradication. Therefore, it would be prudent to continue the current MV vaccination. Ultimately development of novel, safe vaccines which have higher efficacy against the veterinary morbilliviruses is a priority. These would to protect the human population long term against the threat of zoonosis by these veterinary viruses.

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cross species infection; measles; morbillivirus; vaccination; veterinary morbilliviruses; zoonosis

Introduction

Measles virus (MV) is the only human virus within the morbillivirus genus of the *Paramyxoviridae*. The veterinary members are canine distemper virus (CDV), peste des petits ruminants virus (PPRV), Rinderpest Virus (RPV) as well as the marine morbilliviruses phocine distemper virus (PDV), dolphin morbillivirus (DMV) and porpoise morbillivirus (PMV). The World Health Organization (WHO) has set goals towards the eventual elimination of MV in 2020,[1] however, there is potential for new morbillivirus strains from alternative zoonotic reservoirs to move into the ecological niche if MV eradication is achieved and vaccination withdrawn. As found by phylogenetic studies, MV shares closest similarity with the now eradicated RPV.[2,3] This, along with examples of successful cross-species transmission in other RNA viruses, such as Influenza[4] and the SARS and MERS Coronaviruses,[5] highlights the potential risk and possible consequences of cross-species infection and the case for continued MV vaccination or alternatively use of more efficacious specifically designed vaccines.

Measles and morbillivirus diseases

Morbilliviruses have a severe impact on humans and animal species. They confer diseases which have contributed to morbidity and mortality of the population on a global scale. All morbillivirus infections result in lymphopenia and cause the host to be immunosuppressed which allows the invasion of secondary bacterial infections adding to the morbidity and mortality. The characteristic maculopapular rash associated with MV is thought to be caused by the T-cell response to the infection as often individuals with an immunodeficiency do not display this symptom.[6] The virus is transmitted by aerosol droplets and can cause respiratory distress as well as damage to the bronchioles and cilia lining the respiratory tract.[7] It is known to replicate in lymphoid tissues and organs but also affects the skin, lung, conjunctivae and the gastrointestinal (GI) tract. This damage to the GI tract is also seen in other members of the morbillivirus genus, particularly the now eradicated RPV.[2,8] PDV and DMV result in lesions in the lung, lymphoid and central nervous tissues which is similar to symptoms observed with infection by CDV.[9,10] Infection of the central nervous system (CNS) also occurs with morbillivirus species which, in the case of MV, can result in acute post infection encephalitis (1 in 1,000 cases), subacute sclerosing panencephalitis (SSPE) (up to 1 in 10,000 cases) and measles inclusion body encephalitis (MIBE) which occurs in immunosuppressed individuals.[11,12] In contrast to MV, it is usual for the marine morbilliviruses and CDV to infect the CNS in their natural hosts. A rare form of encephalitis which bears resemblance to SSPE, also occurs in mature dogs and is known as 'old dog encephalitis'.[13] It presents symptoms including extensive perivascular cuffs and intranuclear viral

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inclusions which are also associated with SSPE. CDV is most likely the etiological agent in this manifestation of disease as it was detected through molecular assays and immunohistochemical techniques.[13]

There is substantial evidence from both natural and experimental infections[14,15] that morbilliviruses can readily cross species barriers. This is not surprising considering their common origin from a postulated common ancestral virus. MV is thought to have evolved from the now eradicated cattle morbillivirus, RPV, by entering the human population during cattle domestication.[16] Although RPV is now eradicated, this highlights the potential consequences of complete withdrawal of MV vaccination after eradication. Of most concern with regard to zoonosis is the more recently reported fatal infection of primates in Japan and China with strains of CDV which have adapted to this host. In 1989 a case of encephalitis in a Japanese monkey (*Macaca fuscata*) occurred[17] and more recently on a Guangxi breeding farm approximately 10,000 animals were infected with CDV with between 5%–30% mortality. The epidemic was controlled by vaccination.[18] An outbreak of CDV in 20 hand-feeding Rhesus monkeys was later reported in Beijing.[19]

Morbillivirus proteins

One of the major factors in virus adaptation to another species is mutations in the virus protein(s) which allow use of the cell entry receptors of the new host. Morbilliviruses encode six structural and two non-structural proteins. Within the internal helical nucleocapsid, comprised of the nucleoprotein (N), the phosphoprotein (P) and the large protein (L), is the RNA genome which forms a ribonucleoprotein complex together with the RNA dependent RNA polymerase (RdRp).[20] The L protein activates the enzyme RdRp through its interactions with the P and N proteins. This enzyme is responsible for the transcription and replication of the virion genome and also carries out modifications of the mRNAs post-transcriptionally. This enzyme, however, has little proof-reading ability and accounts for the high mutation rate associated with RNA viruses. The non-structural proteins, C and V, are encoded from the P protein gene and have been shown to have roles as interferon antagonists modulating the immune response.[21–23] The virus is enveloped by a lipid bilayer which is formed from the host cell when budding from the plasma membrane occurs. This envelope contains three structural proteins the matrix protein (M) which presents a boundary between the nucleocapsid and the envelope and plays a role in the transcription and budding of the virus[24,25] by interacting with the haemagglutinin (H) protein and the fusion (F) protein.

The H protein is the most important protein in mediating the viral attachment to a specific receptor. The attachment of virus brings about a conformational change in both the H and F proteins. This change in the F protein allows the fusion of the virus with the host cell membrane and the subsequent entry of the nucleocapsid.[26,27] Knowledge of these proteins and their interactions with the host along with sequence similarities among different morbillivirus species provides invaluable information on the mechanisms used by the virus and the risk of cross- species infection.

Morbillivirus cell entry

Several receptors have been identified and characterized in their role in viral entry of MV and the veterinary morbilliviruses. Signalling Lymphocyte Activation Molecule (SLAM), a membrane glycoprotein, has been reported as an MV receptor on immune cells. This molecule has also been shown to act as a receptor for CDV, RPV, PPRV, PDV and DMV.[28–32] It has also been found that these viruses can use SLAM receptors of non-host species albeit with lower efficiencies.[31] The SLAM receptor is selectively expressed on cells of the immune system and can account for the lymphotropism, lymphopenia and immunosuppression of infected individuals.[33,34]

More recently an adherens junction protein, poliovirus-receptor-like-4 (PVRL-4) also known as Nectin-4 was identified as the epithelial receptor for MV.[35,36] The veterinary morbilliviruses have also been shown to use their species specific nectin-4 molecules.[32,37] This receptor is expressed on the basal but not apical surface of epithelial cells and would therefore not allow entry to the respiratory tract. Instead virus entry is considered to occur through SLAM expression on dendritic cells. Nectin-4 would mediate the exit of virus back into the airways and spread of the virus to other individuals via aerosol transmission.[38,39]

Nectin-4 has also been shown to be expressed extensively in canine brain tissue where it could also have a role as a cell entry receptor. Nectin-4 was detected in ependymal cells, epithelia of choroid plexus, meningeal cells, neurons, granular cells, and Purkinje's cells. CDV antigens were detected in these nectin-4-positive cells, further indicating a role of nectin-4 in CDV neurovirulence.[40,41] Studies suggest that the expression pattern of nectin-4 in the CNS differs greatly between dogs and humans. The molecule is difficult to detect in human brain samples[35,36] which could be a factor in why MV CNS infection in humans is a much rarer event then that of CDV or the marine morbilliviruses in their respective hosts.

The major route for morbillivirus entry to the CNS is considered to be across the blood-brain-barrier (BBB) via the cerebral endothelium.[42–44] We have recently shown that nectin-4 cannot be detected by antibody staining and mRNA is only found at very low levels in human brain endothelial cell cultures. However, when these cultures are inoculated with wild type (wt) MV the protein is highly expressed.[45] Therefore, MV may possibly up-regulate the receptor on endothelial cells at the BBB providing a CNS entry mechanism. However, this would not allow spread into other cells in the brain parenchyma such as neurons and oligodendrocytes. Similarly, astrocytes in canine brain do not express nectin-4, although they are frequently infected with CDV.[46] Since astrocytes are negative for SLAM expression, they must express an unidentified CDV receptor, which would also contribute to CDV neurovirulence. This raises the possibility of one or more further receptors for infection of the human and canine CNS by morbilliviruses.

Adaption to receptors in vitro

Other receptors have been found to facilitate morbillivirus cell entry *in vitro*. CD46, a membrane co-factor protein is widely expressed on human and other primate cells.[47,48] While CD46

has been confirmed as a MV receptor it was found to only mediate the entry of adapted vaccine strains *in vitro* but not wt MV. This was shown to be due to amino acid substitutions in the virus H protein. It has been proposed that the lack of CD46 using viruses in vivo may be due to the down regulation of the protein by MV in infected cells following cell entry.[49,50]

Unlike wt MV and wt CDV, we found that wt strains of PDV were able to infect African Green Monkey Vero cells with no prior adaptation. In common with RPV and CDV, we have demonstrated that wt PDV does not utilise CD46 as a receptor.[31] There is evidence to suggest that CDV H protein interacts with an unknown cellular receptor(s) regulated by CD9, a member of the tetraspan transmembrane- protein family.[51] CD46 has been shown to form a complex with CD9, beta1 integrins and the membrane bound form of heparin-binding EGF-like growth factor (pro-HB-EGF).[52,53] We found that infection of wt PDV in Vero cells was inhibited by antibody to HB-EGF and that virus replicated in CHO-pro-HB-EGF cells, indicating use of this molecule as a receptor.[32]

Adaptive mutation to cell entry receptors

We now know that two cell entry receptors are shared across the morbilliviruses, SLAM on immune cells and Nectin-4 on the basal surface of epithelial cells. Furthermore, little or no virus mutation is required for use of these molecules in different species. Langedijk *et al.*[54] identified 11 residues on one side of CDV H protein which contains distinct and overlapping sites that control functional interaction with multiple receptors. Some of these amino acids map onto SLAM and the Nectin-4 binding site. Removal of these sites abrogates binding for MV but not CDV. Sequence analysis of the H gene of three CDV strains adapted to monkeys revealed a glycine (G) and a tyrosine (Y) at amino acid positions 530 and 549 of the partial SLAM-receptor binding region of the CDV H protein. G530 and Y549 are typically found in viral strains obtained from domestic dogs in China rather than wildlife viruses. The three monkey CDV strains possessed E276V, Q392R, D435Y and I542F substitutions, which are unique changes when compared to the other Asia type I lineage strains. In particularly, the I542F substitution falls with the SLAM-binding regions of the H protein. The CDV monkey-BJ01-DV strain was shown to efficiently use monkey- and dog-origin SLAM to infect and replicate in host cells, but further adaptation is likely to be required for efficient replication in host cells expressing the human SLAM receptor.[55]

Vaccination

The MV vaccine is generally given as part of the measles, mumps and rubella (MMR) vaccine, all live attenuated viruses. One dose of MMR vaccine is on average 93% effective for measles while two doses are 97% effective. Both serologic and epidemiologic evidence indicate that vaccine-induced measles immunity appears to be long-term and probably lifelong in most individuals.[56] Many of the attenuated strains in use are derived from the Edmonston strain isolated in 1954, including the Schwartz, the Edmonston-Zagreb, and the Moraten strains. Other strains which are not derived from Edmonston strain include the CAM-70, TD 97, Leningrad-16, and Shanghai 191 (Ji-191) strains. The attenuated production virus is replicated in primary chick embryo or other cell cultures, the virus harvested, clarified, and (alone, or with other antigens) lyophilized (WHO website). The MV vaccine is extremely safe due to the very low risk of reversion but is still unlikely to be acceptable in a measles free world raising the need for alternative approaches. A formalin fixed MV vaccine was used for a period in the 1960s but provided short lived and non-complete immunity with an altered immune response and death of some children following later infection.[57] Vaccines against rinderpest and measles has led to the eradication of the former and the greater control of the latter. Vaccines against PPR and canine distemper have also been generated; however, the diseases still pose a threat to susceptible species.[58]

Canine distemper virus immune-stimulating complexes (iscoms), but not measles virus iscoms were found to protect dogs against CDV infection.[59] In MV vaccinated macaques experimentally infected with CDV the disease was found to be self-limiting. However, virus shedding still occurred from the upper respiratory tract, albeit at lower levels than in non-vaccinated animals.[60] Therefore, to protect the human population against potential zoonotic events by CDV and/or other veterinary morbilliviruses we will need vaccines which unlike the current MV vaccine would prevent virus shedding and hence human to human virus transmission. This is encouraging research into alternative types of vaccines which would be a priority to have in place when MV is eventually eradicated.

Conclusions

The commonality of morbillivirus receptors and the ability of these viruses to adapt to use other host species cells in culture provides a basis for assessing the risk of animal to human transmission of the veterinary morbilliviruses when MV is eventually eradicated. Currently, there is minimal risk of human infection due to the monoserotypic characteristic of morbilliviruses which means that individuals who have received the routine vaccination have a level of protection against other members of this genus. However, the recent adaptation of CDV to non-human primates and associated mutations in the virus H protein increases the possibility of morbillivirus zoonosis, particularly further adaptation of CDV monkey strains to humans. In view of this prospective it would be prudent to continue MV vaccination in the immediate future following eradication. Furthermore, there is a priority to develop of novel, safe vaccines for humans which would more fully protect against the veterinary mobilliviruses by providing sterilizing immunity.

Abbreviations

BBB	blood-brain-barrier
CDV	canine distemper virus
CNS	central nervous system
DMV	dolphin morbillivirus
G	glycine
F	fusion protein
GI	gastrointestinal
H	haemagglutinin protein

L	large protein
M	matrix protein
MIBE	measles inclusion body encephalitis
MMR	measles mumps and rubella
MV	measles virus
N	nucleoprotein
P	phosphoprotein
PDV	phocine distemper virus
PMV	porpoise morbillivirus
PPRV	peste des petits ruminants virus
pro-HB-EGF	membrane bound form of heparin-binding EGF-like growth factor
PVRL-4	poliovirus-receptor-like-4
RdRp	RNA dependent RNA polymerase
RPV	Rinderpest Virus
SLAM	Signalling Lymphocyte Activation Molecule
SSPE	subacute sclerosing panencephalitis
WHO	World Health Organization
wt	with wild type
Y	tyrosine

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